Population-Based Dose Models for Multimedia Chemicals with the Potential for Long Range Transport

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Abstract

This paper is a comparison of methods for characterizing the population-based potential dose to a persistent organic pollutant with the potential for long range transport. If a chemical travels long distances in the environment, more people are exposed to the chemical increasing the potential adverse effects, owing both to the increased number of exposed individuals and to variability in individual susceptibility. Thus a method for calculating the population-based potential dose would be useful for regulators comparing the impact between various chemical emissions. It is unclear what spatial scale and model configuration should be used when calculating the population-based potential dose. Several conceptual models for population-based potential dose are presented and compared. Dose calculations are integrated with the characteristic travel distance of the chemical and population density to determine appropriate methods for evaluating population-based potential dose. A comprehensive multimedia, multipathway exposure model is used to calculate the dose per person. Case studies are presented to illustrate the differences between various calculation methods. We found that if a chemical has a long characteristic travel distance in the environment, it is important to consider exposure to individuals far from the source region when making decisions about the potential hazards from a pollutant.

Keywords: Population Dose, Multimedia Modeling, Long Range Transport, Persistent Organic Pollutants,

Exposure Modeling

Introduction

There are a number of persistent organic pollutants (POPs) that may remain in the environment without transformation for a long time and have the potential to be transported great distances (1-4). If a POP travels a long distance in the environment, more people can be exposed to the chemical and the potential exposure and risk to these individuals needs to be calculated to properly characterize the health impact of the chemical. While public health risk assessment for toxic chemical emissions is well developed for an individual in the vicinity of a contaminated site or point source of emission (5, 6), the risk to individuals farther from the source, and exposed at a lower concentration, is not always considered.

An approach for evaluating the potential dose to the entire population for a chemical release into the environment is desirable because it enables us to differentiate between pollutants that expose many people and pollutants that expose very few people. If a large number of people are exposed to the chemical, there is a higher chance of a member of the population experiencing an adverse effect than if only a small number of people are exposed to the chemical. This is due to both the increased number of exposed individuals and to variability in both intake rates and individual susceptibility (7, 8). As more people are exposed the chemical, the probability of exposing an individual with a low susceptibility increases. Also, if a chemical spreads a long way in the environment, there are likely to be multiple exposures from various sources, increasing the exposure level and hence risk to those individuals exposed to multiple sources (9, 10).

Ideally, the population risk for a uniformly exposed population is calculated by multiplying the risk to a representative individual in that population by the size of the population. However, methods for defining the number of exposed individuals, or zone of impact of a persistent pollutant, are not well defined. At one extreme, we could consider exposure to the nearby urban region, but this would miss exposure to individuals living farther from the point of release. At the other extreme, we could assume the pollutant was evenly spread across the globe, but this would miss the coupling of the higher dose per person and high

population density in the urban region. In reality, when a chemical pollutant is released into the environment, the concentration levels vary spatially. Moreover each pollutant has a different spatial range in the environment, and thus effects different numbers of individuals. Additionally, the population density varies spatially. For these reasons, if we pick a single zone of impact for all chemicals our choice is arbitrary and may not properly characterize the population-based potential dose effectively for all chemicals.

Ideally, we should use a modeling approach that accounts for the chemical specific spatial variability in concentrations and spatial variability in population density. In this paper, we examine various methods for determining the zone of pollutant impact and the corresponding model scale for use in calculating the population-based potential dose. In this paper, several models for calculating population-based potential dose for multimedia pollutants are described and compared. One method accounts for the chemical specific variation in concentration and spatially variable population density. Other modeling approaches use either a chemical specific or fixed area with a single population density. Our goal is to understand how characteristic travel distance influences the relative difference between methods for calculating the population-based potential dose.

Regulators can use population-based potential dose to identify the chemicals with the highest possible adverse health effects, and provide more effective ways to regulate these chemicals. The measure of population-based potential dose can also be used by decision makers in a variety of analyses, including exposure in risk assessments, life cycle analyses, Toxic Release Inventory evaluations, and persistent organic pollutant evaluations.

Many of the persistent pollutants with the potential for long range transport in the environment partition into multiple environmental media. Thus, the potential dose to an individual must account for exposure through multiple exposure pathways, including inhalation, ingestion, and dermal exposure. The model used to calculate exposure in this paper is a comprehensive, multimedia, multipathway model.

There are several examples of calculations of dose to a population in the literature. Thompson and

Evans (11) calculate population risk for inhalation exposure for perchloroethylene from dry cleaners. Smith proposed Exposure Efficiency for establishing total exposure for airborne pollutants and defined it as "the fraction of released material that actually enters someone's breathing zone (12)." Another method for determining the population-based dose for airborne pollutants is the Population Inhalation Transfer Factor defined by Lai et al. as "the pollutant mass inhaled by all members of the exposed population (13)". There is less in the literature on population-based dose for multimedia pollutants. Webster and Connett (14) calculated the population risk for incineration products based on the net deposition to agricultural products.

A study of global chemicals by Travis and Hester (15) calculated the background cancer risk from eleven global pollutants based on measured background concentrations. These results demonstrate a need for research quantifying risk from chemicals with a potential for long range transport. A survey was conducted to determine at what level of risk regulatory action was taken and found that the level was lower for pollutants that exposed a large number of individuals, indicating the regulatory significance of accounting for the entire exposed population (16).

Starting in 1988, the EPA began requiring companies to report the amount of toxic chemicals released from their facilities through the Toxic Release Inventories (TRI) program. Methods for evaluating chemical releases listed in the TRI are being developed, some based on the exposure to the population. The simplest method to compare these releases considers the quantity released and the toxicity of the chemical (17). More advanced methods also include critical factors such as persistence, pollutant fate, or exposure factors (18, 19). It was demonstrated that more advanced methods yield significantly different results than simpler methods. The main drawbacks cited for the advanced methods are that they require more data, which are often unavailable (18), and that increasing the complexity also increases the uncertainty (19).

In life cycle impact analysis (LCIA), analysts are concerned with the total dose to all exposed

individuals from all releases of a chemical in the life cycle of a product. The goal is to use generic release scenarios to develop toxic equivalencies. Chemicals are often evaluated using a fixed area with no advective transport from the region to capture the total chemical release (i.e. wind and water velocities are artificially set to zero)(18, 20). This approach, while conserving mass, can lead to an artificially high dose to each individual in the region and does not consider variations in the population density. Hertwich et al. compared the calculated potential dose with both an open and closed system boundaries for a large number of chemicals (18). They found that an open control volume provides more realistic exposure concentrations, but these concentrations need to be multiplied by a spatial range or number of individuals exposed in order to quantify the cumulative dose to the entire exposed population for the chemical.

This paper presents and compares various conceptual models for calculating population-based potential dose. First, however, the methods used to calculate the dose per person are outlined and the method for calculating the characteristic travel distance for a pollutant in the environment is summarized. Each conceptual model for calculating population-based potential dose utilizes a different area and population density; some incorporating the characteristic travel distance of a particular chemical and the spatially dependent population density into the calculations. We present two case studies, TCDD and benzo[a]pyrene, and compare the parameter uncertainty to the model uncertainty between conceptual models to provide insight on the magnitude of model uncertainty relative to sources of parameter uncertainty. We also use Monte Carlo simulations to develop sets of properties for hypothetical chemicals, that is, a set of chemical "realizations," with each of the properties falling within the plausible range. We calculate the characteristic travel distance and population-based potential dose with each conceptual model to understand the trends between the various calculation methods for the population-based potential dose and characteristic travel distance.

Methods

Dose per person

There are three main steps in an exposure assessment. The first step is to determine the environmental media concentrations for a given source rate, the second step is to determine the exposure media concentrations from the environmental media concentrations, and the third step is to evaluate the dose to an individual based on human activity and contact.

A fugacity-based multimedia model that includes air, two soil compartments, vegetation and surface water is used in this paper (1, 21). The model estimates the steady state concentration in each environmental compartment by balancing gains and losses in each compartment. Most of the processes needed to define the interactions between environmental compartments that define the gains and loses for each compartment are taken from the CalTOX model (22, 23). Exposure media concentrations may differ from the ambient environmental media concentrations and can be calculated from the ambient air, soil, vegetation, and surface water concentrations. The equations relating the exposure media concentrations to the environmental concentrations are taken from the CalTOX model (24).

Humans are exposed to chemicals in the environment through multiple exposure pathways. Exposure and dose are characterized by route of entry as inhalation, ingestion, or dermal uptake. Inhalation exposure includes contact with both indoor and outdoor air. The ingestion pathways include tap water consumption; incidental soil ingestion; and intake of fruits, vegetables, grains, and animal products, such as meat, poultry, eggs, and dairy. The dermal route includes exposure through contaminated water from bathing and recreation, as well as from soil on the skin. The equations used for each exposure pathway are taken from the CalTOX model (24).

Potential dose is calculated from the contact rate with the exposure media and the chemical concentrations in these exposure media (i.e. tap water, indoor air, etc.)[i]. More specifically, the potential dose is calculated from the concentration in the environmental medium, the relationship between the exposure medium concentration and environmental medium concentration, intake rate, body weight,

activity patterns, and exposure duration as (25):

$$ADD = C_{env} \times R \times \frac{CR}{BW} \times \frac{ED \times EF}{AT} \quad (1)$$

where *ADD* is the average daily dose of chemical via exposure route (mg/kg/day), C_{env} is the chemical concentration in the environmental medium (mg/kg), *R* is the ratio of the exposure concentration and the environmental concentration (unitless), *CR* is the contact rate with the exposure media (kg/day), *BW* is the body weight (kg), *ED* is the exposure duration (years), *EF* is the exposure frequency (days/year), and *AT* is the averaging time (days). Exposure duration is assumed to equal averaging time because when calculating the population-based potential dose, the population density is assumed constant in time. The input parameters (e.g. breathing rate, water intake rate, etc.) vary between pathways, thus Equation 1 is specified for each exposure pathway and summed across all exposure pathways. The risk to an individual due to exposure to a carcinogen is calculated by multiplying the *ADD* by a cancer potency factor (CPF).

The average daily dose is also dependent on an individual's activity pattern, such as the percent of each type of food that is grown locally or how much time is spent indoors vs. outdoors. There is variability within a population and additionally, there may be spatial variability between populations. For example, individuals in rural regions may spend more time outdoors than their urban counterparts. Rural individuals might consume a higher percentage of locally grown food because a higher percent of the food is produced in the rural region. In this analysis, we make the assumption that all activity patterns are spatially independent (i.e. the same distributions are used for all individuals). It has been shown that food origin is important in determining the risk (26) and that the assumption of an equal percentage of locally grown food between populations may have a large effect on the total dose to an individual, particularly if the chemical is one that tends to bioconcentrate in the food supply. We feel this assumption is appropriate for this model comparison process.

Estimates of parameter values can rarely be characterized accurately by a single value, due to

both uncertainties in determining a parameter value, variability within the landscape or population, or both. A probability distribution is assigned to each uncertain or variable parameter that has the shape and range conforming to the environmental limits of the selected parameters. Probabilistic distributions for the parameters yield a more comprehensive estimate for risk than point values targeting a hypothetical most sensitive individual based on a set of conservative assumptions (27). The distributions used in this paper were taken from the CalTOX database (28). When calculating a population-based potential dose, we are not concerned with separating the variability between members of the population. Instead, a Monte Carlo simulation varies uncertainty and variability simultaneously to predict the mean value of the population-based potential dose.

Characteristic Travel Distance

A methodology for determining the characteristic travel distance (1) of airborne semi-volatile organic pollutants is used in this paper to characterize the change in environmental concentrations with distance from the source. The concentration in the air is decreased by degradation in air, and transfers to, and subsequent degradation in, the soil, vegetation, and surface water.

The methodology is appropriate for continuous, large non-point atmospheric emissions of organic chemicals, such as collective emissions for a large urban area. The method assumes that the source term is continuous, the system has reached steady state, there is no lateral air dispersion, and the long-term average wind pattern can be represented by an equivalent steady wind rate in one direction.

The concentration profile in any media is approximated using the following equation (1):

$$C_{env}(x,u) = C_{env}(0)e^{-x/CTD} \qquad (2)$$

where x is the distance from the source region (m), and CTD is the characteristic travel distance (m), defined as $u/ k_{effective}$ where u is the average wind velocity in (m/d) and $k_{effective}$ is the effective decay rate,

which is defined as the mass averaged decay rate averaged by the chemical mass in air as explained in Reference 1. At one characteristic travel distance from the source, the concentration in all media is reduced by 63% (i.e. reduced to 1/e of the original concentration).

Population-Based Potential Dose

We use the following equation for the population-based potential dose:

$$Population\ \ Based\ \ Potential\ \ Dose = \iint P(x,y) \times ADD(x,y)\,dx\,dy \qquad (3)$$

where *P* is the population density at location *x, y* (Persons/m^2) and *ADD* is the dose per person at location *x, y* (mg/kg-d). The units of population-based potential dose are Persons·mg/kg-d. If we use a linear CPF, the population risk can be calculated directly from the population exposure. The population-based potential dose multiplied by a linear CPF would yield a predicted expected number of adverse effects. If a non-linear CPF is used, risk must be calculated for each cohort with similar doses before summing across the population.

In Equation 3, both the dose per person and the population density can vary spatially. We must determine the appropriate scale, system boundaries, and population density to use when calculating the population-based potential dose. To evaluate alternative approaches, we consider an idealized environmental model with the source term located in the urban region, where the population density is highest. We assume a steady wind blowing from the urban region toward the suburban and rural regions, which have lower population densities than the urban region. Pollution sources that occur in the suburban and rural regions are excluded in this conceptual model.

In Figure 1a, we present a geometry that accounts for the coupling of the higher population density and higher dose per person in the urban region. We call this the Spatial Model. The concentration is constant in the urban region and decreases exponentially with distance from the source region based on the Characteristic Travel Distance of the chemical due to decay in the environment. This geometry uses a

wind velocity that always travels in a constant direction with no lateral dispersion. Population densities are assumed constant in time in the urban, suburban and rural regions. We believe these simplifications are appropriate because the Spatial Model is designed to compare chemicals, not determine actual risk levels to individuals far from the source region. The equation for the population-based potential dose for the Spatial Model is:

$$Population\ \ Based\ Potential\ Dose = P_U \times ADD \times w_y w_x + \int_{w_x}^{w_x'} P_{SU} \times ADD \times e^{-(x-w_x)/CTD} \times w_y\, dx + \int_{w_x'}^{\infty} P_R \times ADD \times e^{-(x-w_x)/CTD} \times w_y\, dx \qquad (4)$$

the subscripts *U, SU, and R* refer to urban, suburban, and rural, respectively, w_x is the width of the urban region in the x direction (m), and w_y is the width of the urban region in the y direction (m) and w_x' is the distance at which the population density changes from suburban to rural equal to $(A_u+A_{su})/w_y$ where A_u and A_{su} are the areas of a representative urban region and suburban region, respectively.

The ADD is calculated in an open region (i.e. wind flows out of the region as in Figure 1d) the size of a representative urban region. The size and population densities of the urban, suburban, and rural regions are representative, with representative values listed in Table I. The width and length of the urban region, w_x and w_y, are both taken as equal to the square root of a representative urban area.

Next, we present four simplified conceptual models. In each model, a constant population density and an equal dose per person are used to calculate the population-based potential dose. Under these conditions, the population-based potential dose equation can be simplified to:

$$Population\ \ Based\ Potential\ Dose = P \times ADD \times A \qquad (5)$$

where A is the area of the model region (m^2).

For the Chemical Specific Model, we use the characteristic travel distance from the previous

section to determine an appropriate model area for each chemical. In Figure 1b, we illustrate that the region size will increase as the characteristic travel distance of a chemical pollutant increases. The length of each side of the model region is 2.3 times the characteristic travel distance (the distance at which the concentration is reduced to 10% of the original concentration using Equation 2). Closed system boundaries are used to account for the entire chemical mass released into the system (i.e. there is no advection by air of chemical out of the system). We determine the population density in this region by considering what fraction of the area is urban, suburban, and rural and averaging across the region. The *ADD* is calculated for an individual within the Chemical Specific Model area and used for the entire population.

In Figure 1c, we illustrate the Closed Urban Model, a model configuration sometimes used in life cycle analyses. The model area is typical of an urban region. This model accounts for the entire chemical pollutant mass released into a region by closing the model system boundaries to advective flows of air. The water still flows out of the system, preventing a buildup of chemical in the water due to runoff and erosion. A representative urban population density is used in the calculation. The *ADD* is calculated for an individual in the Closed Urban Model area and is used for the entire population.

The Open Urban Model is illustrated in Figure 1d, a scenario typical of a traditional risk assessment. The wind advects air and the associated chemical out of the system. We do not account for human exposure to this mass of chemical. We calculate the *ADD* to an individual in the open urban region and multiply the ADD by the size and population density typical of an urban region. We refer to the population-based potential dose calculated with this method as the Open Urban Model.

We also use the *ADD* calculated for an open urban region (Figure 1d) to calculate the population-based potential dose for the Scaled Model. In the Scaled Model, we use the dose per person as calculated in the Figure 1d and scale the total exposure by the Characteristic Travel Distance for each chemical. We do this by using the area and population density used in the Chemical Specific Model in Equation 5.

Additionally, the population-based potential dose can be calculated for each model using a

background population density. We use the same model boundaries, but with a background population density. For reference, each approach is listed in Table II with the corresponding population densities, area, and figure number appropriate for calculating the average daily dose per person.

Case Studies

The primary goal of the case studies is to compare the calculated population-based potential dose for each of the conceptual models. This comparison focuses on evaluating the following two questions:

Are the differences in population-based potential dose between calculation models significant compared to other sources of uncertainty?

How do these trends change as the characteristic travel distance increases?

To answer the first question, we compare the models by calculating the population-based potential dose and associated parameter uncertainty using each of the conceptual models. We do this for two chemicals, one with a long characteristic travel distance and one with a short characteristic travel distance.

First we examine 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), which has a relatively long characteristic travel distance, on the order of 600 km (1). TCDD is typically released into the air as a by-product from incineration, combustion of fossil fuels, and industrial processes in urban areas but often contaminates suburban and rural sites as well, consistent with the long travel distance in the environment (29-31). Airborne TCDD is found in both the gaseous and particulate phases. In the vapor phase of the atmosphere, reaction with OH radicals is the dominant degradation pathway (32, 33). TCDD on particles has negligible degradation (34). The primary degradation process for TCDD in vegetation is the reductive dehalogenation by sunlight that requires proton donors. Because the lipids in plants are rich sources of proton donors, we expect higher degradation rates in vegetation relative to air (35-37). There is limited degradation in soil for this compound (37-39).

For comparison, we analyze benzo[a]pyrene, which has a relatively short characteristic travel

distance, on the order of 30 km. Benzo[a]pyrene is a polyaromatic hydrocarbon that decays rapidly in air and tends to favor the lipid phases of the environment. Benzo[a]pyrene is a byproduct of combustion and other industrial processes. This chemical is found at much higher concentrations in urban regions, indicating that it does not travel a long way in the environment, consistent with the calculated characteristic travel distance (40). At present, there is no information on the degradation rate of benzo[a]pyrene in vegetation and we assumed a decay rate equal to that in surface soil. The representative values used for all of the chemical properties for both compounds are listed in Table III.

We calculate the population-based potential dose for each chemical for each of the 5 models. A gram per day of each chemical is emitted to the system in each calculation. The parameter uncertainties are propagated through the calculations of the population-based potential dose using 5000 Monte Carlo simulations, implemented with Crystal Ball software (41). The nominal value of the characteristic travel distance was used in the Spatial, Chemical Specific, and Scaled uncertainty calculations.

To answer the second question and understand the relationship between the characteristic travel distance and the differences between the population-based potential dose models, we calculate the characteristic travel distance and the population-based potential dose for 5,000 chemical realizations for each of the 5 conceptual models. The 5,000 sets of properties for the chemical realizations are developed using the Monte Carlo package Crystal Ball (41) such that each of the properties falls within the plausible range. The chemical parameter value ranges used in the simulations are presented in Table IV. Also listed are examples of chemicals with property values near the minimum and maximum of each range. Most of the distributions are log-uniform (a uniform distribution in logarithmic space), yielding the same number of simulations in each decade of the range[ii].

Results

The cumulative distributions for the population-based potential dose to TCDD using each of the

calculation models are shown in Figure 2a. For TCDD the difference between model results is significant compared to parameter uncertainty, and thus it is important to consider what model to use when calculating the population-based potential dose. This is likely to be the case for other chemicals with a long characteristic travel distance.

We learn from Figure 2a that the Spatial Model (Figure 1a) falls in the middle of the five models. This model accounts for the coupling of the high dose per person in the urban region and accounts for all of the pollutant released to the system. The Closed Urban Model (Figure 1c) and the Scaled Model (derived from Figure 1d) result in the higher population-based potential doses. The Closed Urban Model predicts a higher overall dose because it does not account for the decreasing population density as the chemical travels away from the site. In effect, this model assumes no spatial variation in population density, instead using a constant urban population density. The Scaled Model results in a high dose because a large number of individuals are exposed at the concentration level calculated for the Open Urban Model, not accounting for the decrease in concentration as you move away from the source. The Chemical Specific Model (Figure 1b) and the Open Urban Model (derived from Figure 1d) predict lower doses. The Open Urban Model predicts a smaller dose because much of the chemical is advected by wind out of the model system, and there is no exposure to what is advected out of the system. The Chemical Specific Model predicts a smaller dose because it does not account for the coupling of high dose per person and high population density in the urban region. Instead, the population density and dose per person are evenly distributed across the region.

The comparison between parameter uncertainty and model uncertainty is also completed for a chemical with a short characteristic travel distance, in this case, benzo[a]pyrene. The cumulative distributions using each conceptual model are plotted in Figure 2b. For benzo[a]pyrene, the methods yield similar results thus the difference among models is small compared to the parameter uncertainties. The results for benzo[a]pyrene indicate that the model choice is not important when calculating the population-

based potential dose. This may be the result for other chemicals with a short characteristic travel distance. This is in contrast to the conclusions drawn for TCDD. The notable difference between these results leads us to consider the effect of characteristic travel distance on the difference between the calculated population-based potential dose between conceptual models.

To understand the relationship between the characteristic travel distance and the differences between the population-based potential dose models, the characteristic travel distance and the population-based potential dose for 5,000 chemical realizations for each of the 5 conceptual models was calculated. We calculate the ratio of the calculated dose for each model setup to the calculated dose using the Spatial Model. Figure 3 is a plot of these ratios for each model configuration. The results are normalized to the Spatial Model, which accounts for the spatial variation in the dose and the population density. This model is used as a benchmark because it closely approximates the idealized conceptual model and because the results from this model fall between the other distributions. The normalized results have been plotted versus the characteristic travel distance to show trends with increasing characteristic travel distance.

The Scaled Model, which assumes the population density and area used in the Chemical Specific Model with the dose per person found in the Open Urban Model, results in a much smaller population-based potential dose than the Spatial Model for chemicals with a short characteristic travel distance. At the scale of the urban area, the Scaled Model and Spatial Model are equivalent and thus result in equal values for the population-based potential dose. For chemicals with a greater characteristic travel distance, the calculated population-based potential dose using the Spatial Model increases rapidly and provides an upper bound on the possible population-based potential dose. By exposing a larger number of people to the concentrations present in the urban region, we are effectively increasing the size of the source.

We next consider the Closed Urban Model , which predicts a higher population-based potential dose than the Spatial Model for long characteristic travel distances. This result occurs because the model does not account for the decreasing population density as one moves away from the site. The ratio

between the Closed Urban Model and Spatial Model approaches a value of 45, the ratio between the urban and background population density. This occurs when the majority of the exposed population resides in a region with the background population density. This model also tends to yield a higher dose per person to the individuals in the urban region as there is no advection from the region and thus should not be used for evaluating the risk to an individual within the region.

The Chemical Specific Model predicts the same population-based potential dose as the Spatial Model for chemicals with a Characteristic Travel Distance less than the scale of the urban region. As the Characteristic Travel Distance increases, the model predicts a smaller population-based potential dose than the Spatial Model. This is because the high population density with the higher environmental concentrations is not linked as in the Spatial Model. As the characteristic travel distance continues to increase, the pollutant becomes a global pollutant, and thus the effects of the population density become negligible because the majority of the exposed individuals are exposed at a background concentration. Hence, calculations made with the two models are equal for extremely large values of the characteristic travel distance.

The Open Urban Model predicts the same population-based potential dose for chemicals with a characteristic travel distance less than or slightly greater than the urban scale because at this scale, very little of the chemical is being advected out of the Open Urban Model system boundaries. As the characteristic travel distance of the chemical increases, a larger proportion of the chemical is advected out of the Open Urban Model relative to the amount of chemical decayed within the system. Individuals are still exposed to the chemical that is advected out of the system but the resulting exposure is not accounted for, resulting in a lower value for the population-based potential dose when compared to the other models.

From this evaluation we conclude that for chemicals with a characteristic travel distance less than or equal to an urban scale, the model choice is not as important for determining the population-based potential dose as for chemicals with a long characteristic travel distance. With the exception of the Scaled

Model, all of the models yield similar results at this scale. For chemicals with a longer characteristic travel distance, this choice becomes more important.

Similar calculations were made assuming a constant background population. The ratio of the Open Urban Model, Closed Urban Model, and Scaled Model to the Chemical Specific Model, each using a background population density are plotted in Figure 4. With a constant population density, the Closed Urban Model and Chemical Specific Model give equivalent results as they are both closed systems. As in the previous comparison, the population-based potential dose for the Open Urban Model decreases relative to the other models as the characteristic travel distance increases. In contrast, the Scaled Model has an increasing population-based potential dose relative to the other models with an increasing characteristic travel distance. This is because individuals further from the urban center receive the same dose per person as individuals in the urban region.

To quantify the difference between model calculations using a background population density and a spatially varying population density, the ratio between the calculated population-based potential dose using the Spatial Model and the Chemical Specific Model is also plotted in Figure 4. The ratio between the methods begins at 45, the value of the ratio between urban and background population densities, and approach the same answer as the characteristic travel distance increases.

Discussion

We have presented various conceptual models for calculating population-based potential dose. For chemicals whose characteristic travel distance is on the same scale as the size of a typical urban center (less than 200 km), sources of parameter uncertainty in determining the population-based potential dose are greater than the differences between the model results. In fact, for all these chemicals, all of the models yield the same result with the exception of the Spatial Model. The model choice between the remaining configurations is not relevant.

For chemicals with an intermediate travel distance, the choice of a model is important and should be based on the use pattern of the chemical, and the population density of the population likely to be exposed to that chemical based on the use pattern. As the characteristic travel distance increases, the differences between calculation methods can be on the same order of magnitude as the other sources of uncertainty in the calculation. This result is chemical specific, but was the case for TCDD and would be for chemicals with a similar range of parameter uncertainties. If the pollutant is used in an urban region, the Spatial Model is a good choice as it accounts for the coupling of the higher dose per person and the higher population density in the urban region while also accounting for exposures farther from the use area.

If a chemical has a very long characteristic travel distance (greater than1000 km), it is a global pollutant, and it is appropriate to use a Closed Urban Model with a background population density, the Chemical Specific Model, or the Spatial Model. For global pollutants, one also needs to consider that the global food supply will be effected by the elevated concentrations of the chemical and adjust the calculations to account for this. One also needs to realize that each individual will be exposed to the pollutant from multiple sources.

In all of the calculations, several assumptions were made, including: spatially independent exposure parameters, no dispersion of airborne chemicals, all sources located in urban regions, a uniform population density in each of the three categories, each assumed to be in a uniform geographical pattern. We have not evaluated the effects of these assumptions on the reliability of the results because this model is intended for screening level purposes in order to compare chemicals, not to determine the level of risk.

When making choices among alternative chemicals for use in a certain process, evaluating a new chemical upon its introduction to commerce, or deciding if one should further regulate a chemical presently in use, we may want to determine the population-based potential dose to that chemical per unit release. If a chemical has a short characteristic travel distance in the environment it is sufficient to calculate the exposure to individuals in the surrounding region. However, if a chemicals has a long characteristic travel

distance in the environment, we must also consider the exposure to a population far from the site.

For chemicals with an intermediate or long characteristic travel distance in the environment, we need to determine the appropriate system boundaries for calculating this measure. We compared five conceptual models for calculating the population-based potential dose and found that these chemicals, the calculated exposure varied between calculation methods. If the chemical is used in the urban region and has an intermediate travel distance, we recommend the spatial model. One must consider the use pattern of the chemical being evaluated when determining the appropriate model. We recommend that if a chemical has a long characteristic travel distance the model should be selected carefully to make sure the exposure is distributed in a manner consistent with the model assumptions.

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Footnotes

[i] Potential dose, or intake dose, has been defined as the amount of chemical that passes into an individual while the actual dose quantifies the amount of chemical that is absorbed into an individual (e.g. the amount of chemical in the air an individual breathes is the potential dose while the actual dose is the portion of that air that passes into the lung tissue)(42). Ideally, risk should be based on the actual dose, but often the potential dose is assumed to equal the dose, an assumption also made in this paper.

[ii] This method was used to generate chemical realizations have been generated in this manor for understanding what properties are likely to lead to a persistent pollutant in Reference (43) and more information on the process can be obtained from this reference.

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Table I: Representative Population Densities and Areas

	Population Density	Area
	Persons/m^2	m^2
Urban Regions	$P_U=3.52\times10^{-3}$	$A_U=2.56\times10^{9}$
Suburban Regions	$P_{SU}=5.86\times10^{-4}$	$A_{SU}=5.12\times10^{9}$
Background	$P_B=7.81\times10^{-5}$	

Table II: List of all conceptual population-based dose models

Conceptual Model Name	Figure	Model Area Used	Population Density	Calculation Method
Spatial Model	1a	a	a	$PBPD = P_U \times ADD \times w_y w_x + \int_{w_x}^{w_x'} P_S \times ADD \times e^{-(x-w_x)/CTD} \times w_y \, dx + \int_{w_x'}^{\infty} P_B \times ADD \times e^{-(x-w_x)/CTD} \times w_y \, dx$
Chemical Specific Model	1b	$(2.3 \times L)^2$	P_S	$ADD \times (2.3 \times L)^2 \times P_S$
Chemical Specific Model Background Population	1b	$(2.3 \times L)^2$	P_B	$ADD \times (2.3 \times L)^2 \times P_B$
Closed Urban Model	1c	A_U	P_U	$ADD \times A_U \times P_U$
Closed Urban Model Background Population	1c	A_U	P_B	$ADD \times A_U \times P_B$
Open Urban Model	1d	A_U	P_U	$ADD \times A_U \times P_U$
Open Urban Model Background Population	1d	A_U	P_B	$ADD \times A_U \times P_B$
Scaled Model	1d	A_U	P_S	$ADD \times (2.3 \times L)^2 \times P_S$
Scaled Model Background Population	1d	A_U	P_B	$ADD \times (2.3 \times L)^2 \times P_B$

$$P_S = \begin{cases} P_U \text{ if } (2.3 \times L)^2 < A_U \\ (P_U \times A_U + P_{SU} \times ((2.3 \times L)^2 - A_U)) / (2.3 \times L)^2 \text{ if } A_U < (2.3 \times L)^2 < A_{SU} \\ (P_U \times A_U + P_{SU} \times A_{SU} + P_B \times ((2.3 \times L)^2 - A_{SU})) / (2.3 \times L)^2 \text{ if } (2.3 \times L)^2 > A_{SU} \end{cases}$$

a) accounted for in calculation method

Table III: Representative Chemical Properties Used in the Case Study

Chemical or Landscape Property	Notation	Mean Value	Coefficient of Variation	Mean Value	Coefficient of Variation
		TCDD	TCDD	B[a]P	B[a]P
molecular weight (g/mol)	*MW*	322	0.01	252	0.01
octanol-water partition coefficient	K_{ow}	5.70×10^{6}	1	2.20×10^{6}	0.72
melting point (K)	*Tm*	578	0.01	451	0.028
vapor pressure in (Pa)	*VP*	1.0×10^{7}	2	7.13×10^{-7}	.07
Henry's law constant (Pa-m^3/mol)	*H*	3.75	1.5	0.092	1
diffusion coefficient, pure air (m^2/s)	D_{air}	4.86×10^{-6}	0.1	0.44	0.08
diffusion coefficient; pure water (m^2/s)	D_{water}	5.90×10^{-10}	0.1	5.3×10^{-5}	0.25
organic carbon partition coefficient	K_{oc}	5.40×10^{6}	0.1	2.49×10^{6}	0.9
biotransfer factor, plant/air (m^3[a]/kg[pFM])	K_{pa}	2.5×10^{4}	0.85	5.92×10^{5}	14
decay rate in air (1/s)	k_a	8.0×10^{-7}	1.5	1.3×10^{-4}	1
decay rate in surface soil (1/s)	k_g	2.2×10^{-8}	1.2	3.5×10^{-8}	1.1
decay rate in root-zone soil (1/s)	k_s	2.1×10^{-10}	1.7	3.5×10^{-8}	1.2
decay rate in surface water (1/s)	k_w	1.6×10^{-3}	1.2	3.4×10^{-6}	1.2
decay rate in vegetation (1/s)	k_p	1.3×10^{-6}	3.0	3.5×10^{-8}	3.0

Table IV: Distributions used for calculating chemical realizations

Property	Symbol	Distribu-tion type	Lower End	Upper End	Example of Chemical with Property at Lower End of Range	Example with Property at Upper End of Range
Henry's law constant (Pa-m^3/mol)	K_H	log uniform	$1x10^{-3}$	$1x10^{5}$	Phenol	Nitrogen gas
octanol-water partition coefficient	k_{ow}	log uniform	1	$1x10^{9}$	Butanol, Methylchloride	Di-n-octyl-phthalate
decay rate in air (1/day)	k_a	log uniform	$4x10^{-4}$	$1x10^{2}$	Toxiphane, Bromodichloromethane	Benzo(a)Pyrene
decay rate in water (1/day)	k_w	log uniform	$1x10^{-5}$	$1x10^{2}$	hexachloroethane	Pyrene
decay rate in soil (1/day)	k_s	log uniform	$1x10^{-5}$	$1x10^{2}$	PCB	Anthracene
vapor pressure in (Pa)	VP	log uniform	$1x10^{-6}$	$1x10^{5}$	Chrysene, TCDD	Atmospheric Pressure
melting point (K)	Tm	uniform	100	600	Vinyl Chloride	Chrysene, beta – HCH, TCDD
Diffusion coefficient in pure air (m^2/s)	D_{air}	uniform	.2	1.7	Hexachloroethane	2,4 – Dinitrotoluene
Diffusion coefficient; pure water (m^2/s)	D_{water}	uniform	3.00E-05	1.00E-4	Endrin	Vinyl Chloride

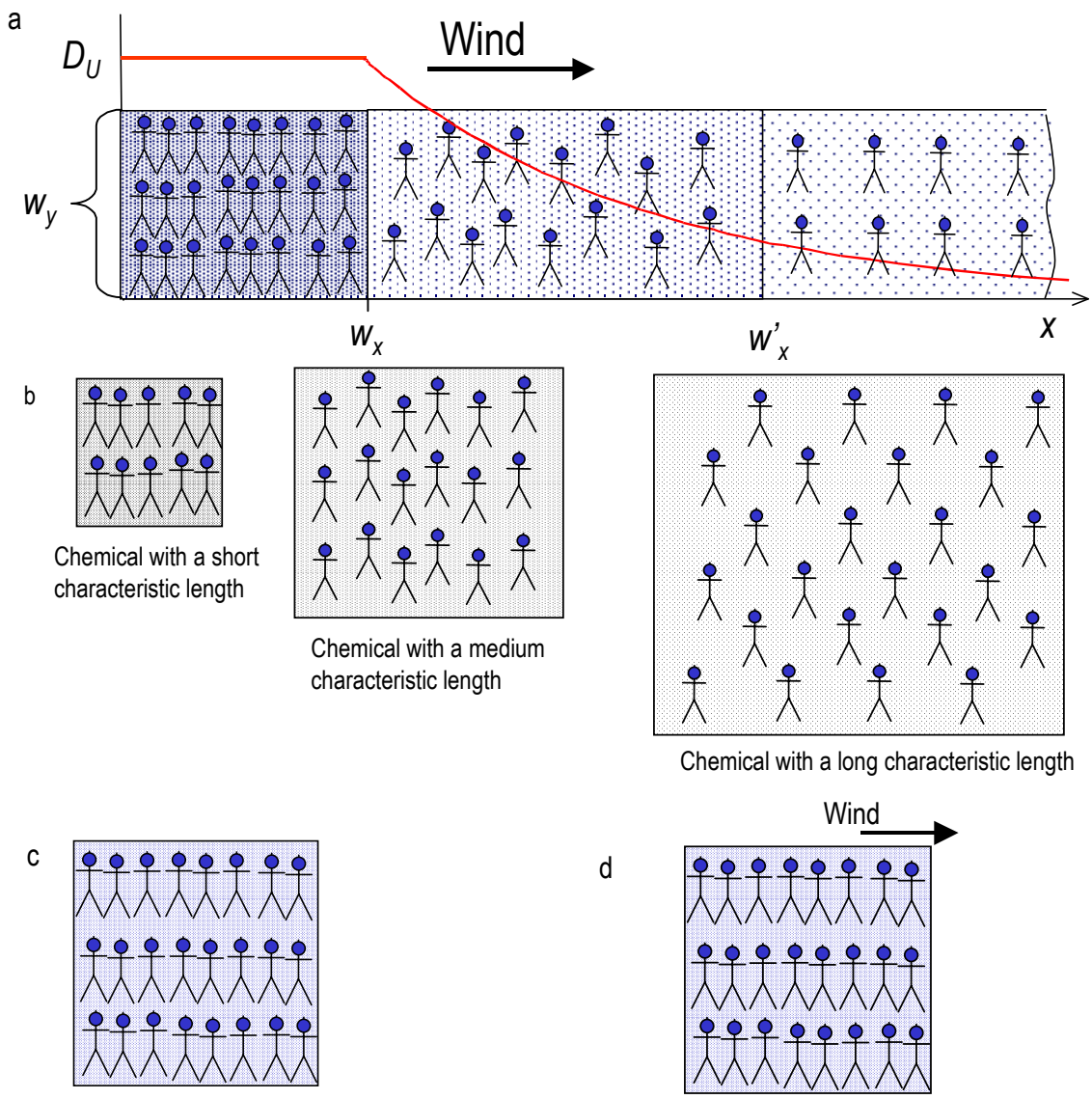

Figure 1: Models used for calculating population-based potential dose
1a: Spatial Model, 1b: Chemical Specific Model, 1c: Closed Urban Model
1d: Open Urban Model, used also for the Scaled Model

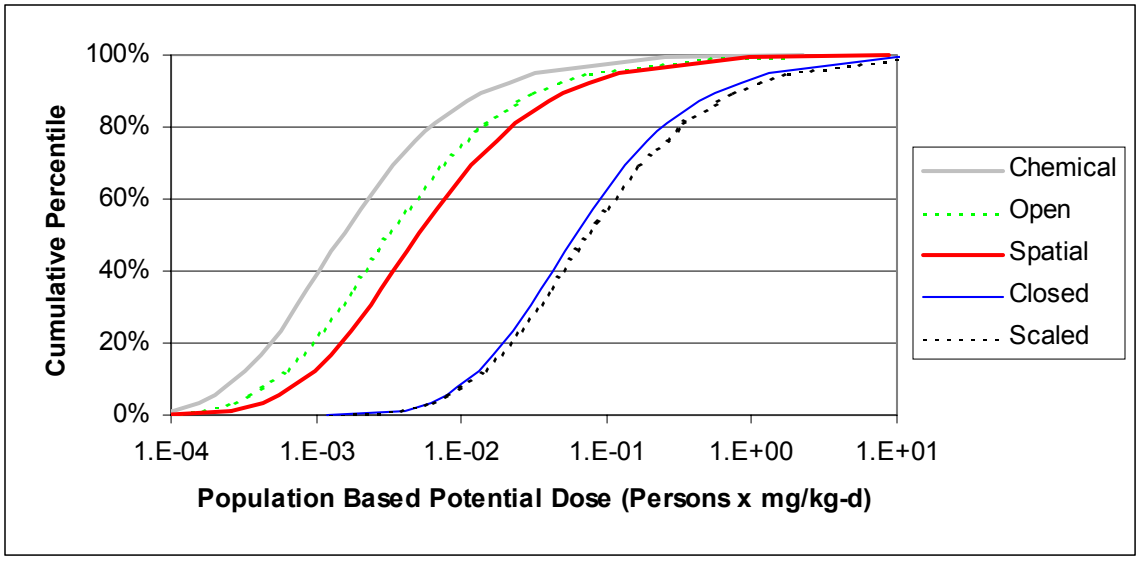

Figure 2a: Cumulative percentile distribution of population-based dose for each calculation method for TCDD (long characteristic travel distance).

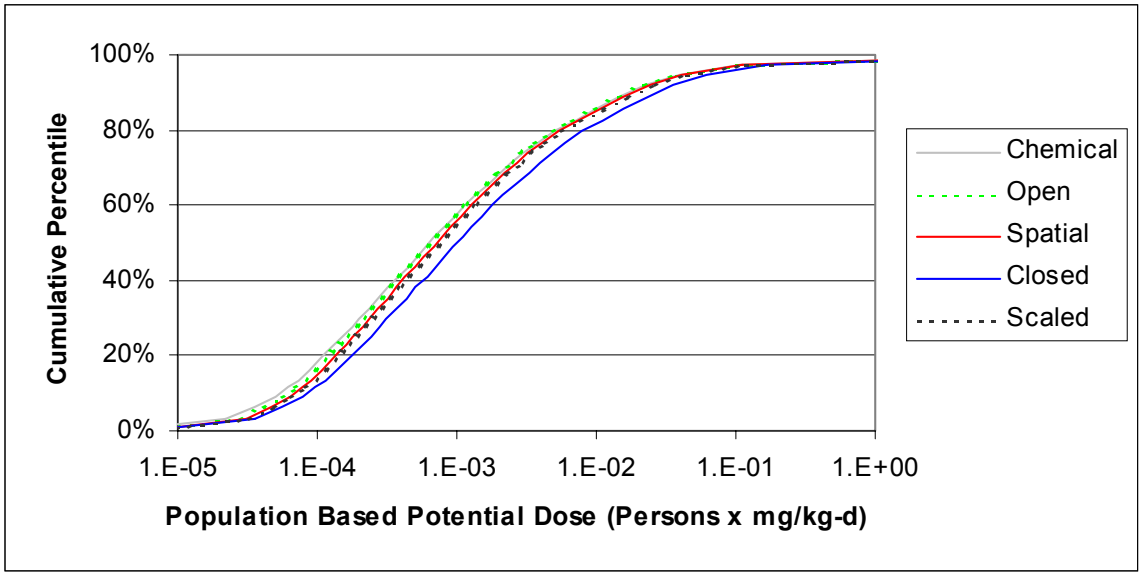

Figure 2b: Cumulative percentile distribution of population-based dose for each calculation method for benzo[a]pyrene (short characteristic travel distance).

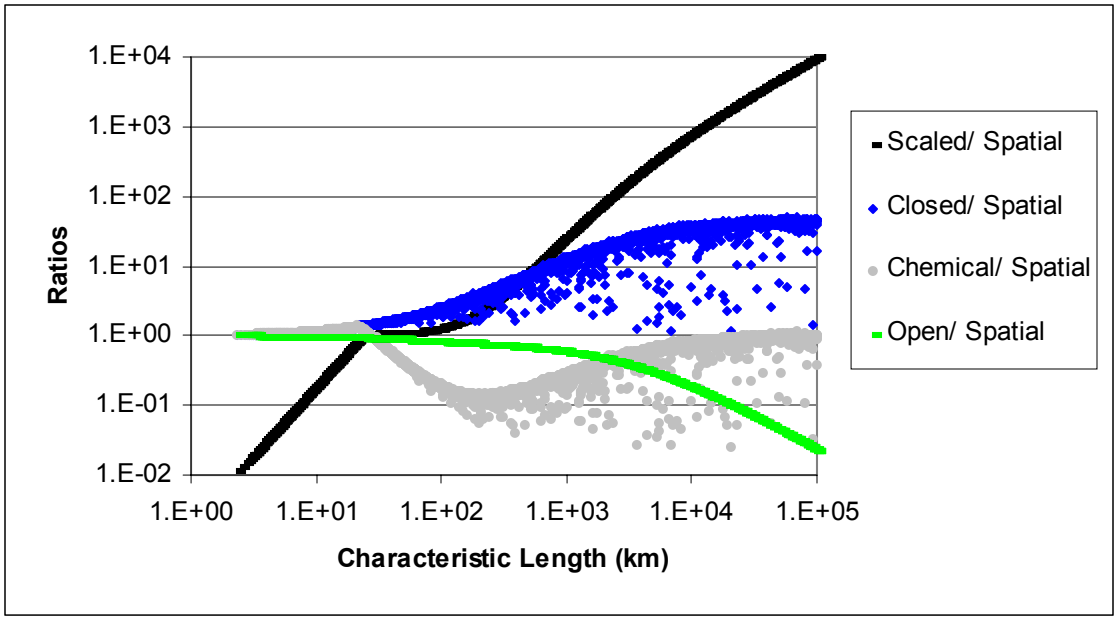

Figure 3: Ratios of population-based dose for each calculation method over the population-based dose for the Spatial Model vs. characteristic travel distance of the chemical for 5000 simulated chemicals.

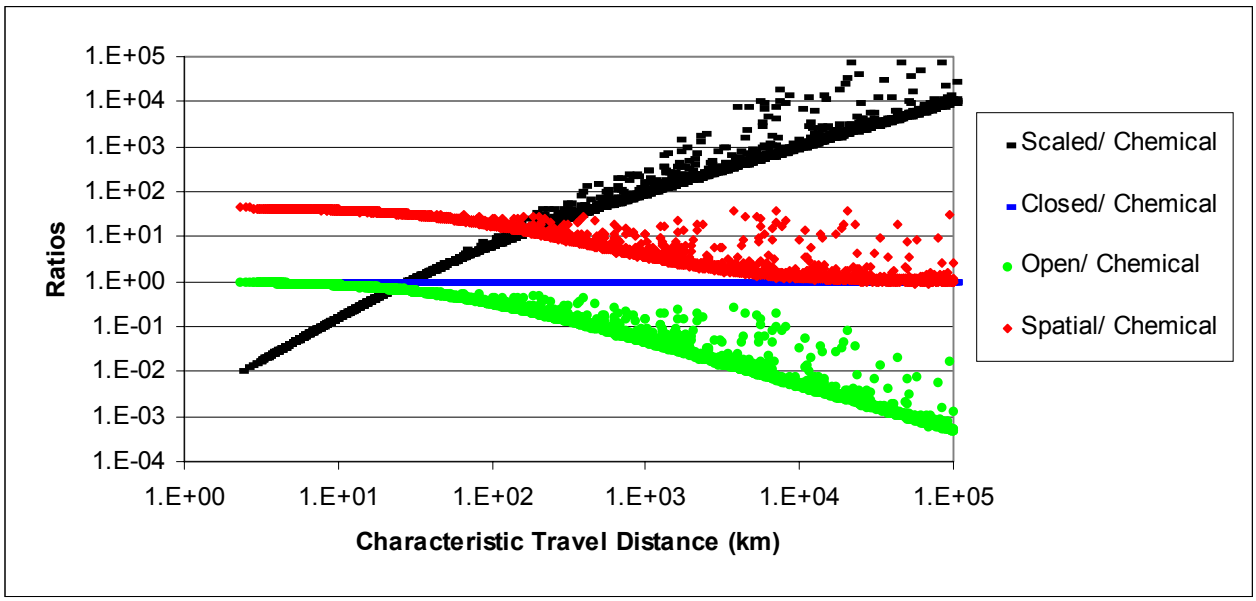

Figure 4: Using a background population density in each model, a comparison of the ratio of population-based dose for the Scaled Model, Closed Urban Model, and Open Urban Model to the population-based dose for the Chemical Specific Model versus the characteristic travel distance. Also, the ratio of the population-based dose for the Spatial Model with varying population density to the population-based dose for the Chemical Specific Model with a background population density versus the characteristic travel distance.